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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Apr 08	"Ask CAS" for self-help around the clock
NEWS 3	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 4	Apr 09	ZDB will be removed from STN
NEWS 5	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 6	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 7	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 8	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 9	Jun 03	New e-mail delivery for search results now available
NEWS 10	Jun 10	MEDLINE Reload
NEWS 11	Jun 10	PCTFULL has been reloaded
NEWS 12	Jul 02	FOREGE no longer contains STANDARDS file segment
NEWS 13	Jul 22	USAN to be reloaded July 28, 2002; saved answer sets no longer valid
NEWS 14	Jul 29	Enhanced polymer searching in REGISTRY
NEWS 15	Jul 30	NETFIRST to be removed from STN
NEWS 16	Aug 08	CANCERLIT reload
NEWS 17	Aug 08	PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 18	Aug 08	NTIS has been reloaded and enhanced
NEWS 19	Aug 19	Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN
NEWS 20	Aug 19	IFIPAT, IFICDB, and IFIUDB have been reloaded
NEWS 21	Aug 19	The MEDLINE file segment of TOXCENTER has been reloaded
NEWS 22	Aug 26	Sequence searching in REGISTRY enhanced
NEWS 23	Sep 03	JAPIO has been reloaded and enhanced
NEWS 24	Sep 16	Experimental properties added to the REGISTRY file
NEWS 25	Sep 16	Indexing added to some pre-1967 records in CA/CAPLUS
NEWS 26	Sep 16	CA Section Thesaurus available in CAPLUS and CA

NEWS EXPRESS	February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
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FILE 'HOME' ENTERED AT 13:21:20 ON 01 OCT 2002

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=> s etanercept

L1 1544 ETANERCEPT

=> s p55tnfr:fc

L2 1 P55TNFR:FC

=> s friedrichs g /au

L3 21 FRIEDRICHS G

=> s swillo r /au

L4 8 SWILLO R

=> s l3 or l4

L5 29 L3 OR L4

=> dup rem l5

PROCESSING COMPLETED FOR L5
L6 22 DUP REM L5 (7 DUPLICATES REMOVED)

=> s l1 and l6

L7 0 L1 AND L6

=> d l6 total ibib kwic

L6 ANSWER 1 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:302106 CAPLUS
DOCUMENT NUMBER: 135:113676
TITLE: Nonequilibrium excitation of C2 radicals during the thermal decomposition of C3O2 behind shock waves
AUTHOR(S): Deppe, J.; Emelianov, A.; Eremin, A.; **Friedrichs, G.**; Shumova, V.; Wagner, H. Gg.; Zaslonko, I.
CORPORATE SOURCE: Institut fur Physikalische Chemie, Universitat Gottingen, Gottingen, D-37077, Germany
SOURCE: Zeitschrift fuer Physikalische Chemie (Muenchen, Germany) (2001), 215(3), 417-425
CODEN: ZPCFAX; ISSN: 0044-3336
PUBLISHER: R. Oldenbourg Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT
AU Deppe, J.; Emelianov, A.; Eremin, A.; **Friedrichs, G.**; Shumova, V.; Wagner, H. Gg.; Zaslonko, I.

L6 ANSWER 2 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:682740 CAPLUS
DOCUMENT NUMBER: 129:266133
TITLE: The thermal decomposition of NH2 and NH radicals
AUTHOR(S): Deppe, J.; **Friedrichs, G.**; Ibrahim, A.; Roemming, H.-J.; Wagner, H. G.
CORPORATE SOURCE: Institut Physikalische Chemie, Universitaet Goettingen, Goettingen, D-37077, Germany
SOURCE: Berichte der Bunsen-Gesellschaft (1998), 102(10), 1474-1485
CODEN: BBPCAX; ISSN: 0940-483X
PUBLISHER: Wiley-VCH Verlag GmbH
DOCUMENT TYPE: Journal
LANGUAGE: English
AU Deppe, J.; **Friedrichs, G.**; Ibrahim, A.; Roemming, H.-J.; Wagner, H. G.

L6 ANSWER 3 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:2651 CAPLUS
DOCUMENT NUMBER: 128:66919
TITLE: Investigation of the thermal decay of carbon suboxide
AUTHOR(S): **Friedrichs, G.**; Wagner, H. G.
CORPORATE SOURCE: Institut Physikalische Chemie, Universitaet Goettingen, Goettingen, D-37077, Germany
SOURCE: Zeitschrift fuer Physikalische Chemie (Munich) (1998), 203(1/2), 1-14
CODEN: ZPCFAX; ISSN: 0044-3336
PUBLISHER: R. Oldenbourg Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English
AU **Friedrichs, G.**; Wagner, H. G.

L6 ANSWER 4 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:251454 CAPLUS
DOCUMENT NUMBER: 124:347627
TITLE: Phenomena related to the storage of natural gas in underground caverns
AUTHOR(S): Gregorowicz, J.; Peters, C. J.; de Swaan Arons, J.;

Friedrichs, G.; Jaeschke, M.
CORPORATE SOURCE: Delft University of Technology, Department of Chemical
Engineering and Materials Science, Laboratory of Applied Thermodynamics and Phase Equilibria, Julianalaan 136, BL Delft, 2628, Neth.
SOURCE: Fluid Phase Equilibria (1996), 117(1-2), 249-56
CODEN: FPEQDT; ISSN: 0378-3812
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AU Gregorowicz, J.; Peters, C. J.; de Swaan Arons, J.; **Friedrichs, G.** ; Jaeschke, M.

L6 ANSWER 5 OF 22 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1991:40074 BIOSIS
DOCUMENT NUMBER: BR40:17054
TITLE: EFFECTS OF 5 LIPOXYGENASE OR PAF ANTAGONISM AND ADENOSINE AGONISTS IN AN IN-VITRO NEUTROPHIL-DEPENDENT MODEL OF REPERFUSION INJURY.
AUTHOR(S): BARRETT J A; **SWILLO R**; WOLTMANN R; PERRONE M H
CORPORATE SOURCE: RORER CENTRAL RES., KING OF PRUSSIA, PA.
SOURCE: 63RD SCIENTIFIC SESSIONS OF THE AMERICAN HEART ASSOCIATION,
DALLAS, TEXAS, USA, NOVEMBER 12-15, 1990. CIRCULATION, (1990) 82 (4 SUPPL 3), III702.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English
AU BARRETT J A; **SWILLO R**; WOLTMANN R; PERRONE M H

L6 ANSWER 6 OF 22 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
ACCESSION NUMBER: 1988:372562 BIOSIS
DOCUMENT NUMBER: BA86:56472
TITLE: TREATMENT OF A SEVERE PRESUMPTIVE HERPESVIRUS ENCEPHALITIS WITH A COMBINATION OF ACYCLOVIR AND INTERFERON FOLLOWED BY COMPLETE RESTITUTION.
AUTHOR(S): SCHMIDT J; **FRIEDRICHS G**; HEINMUELLER D; WACH J
CORPORATE SOURCE: MEDIZINISCHE KLINIKEN I UND II, NEUROL. KLINIK, BUERGERHOSP. STUTTGART, 7000 STUTTGART.
SOURCE: INTENSIVMEDIZIN, (1988) 25 (3), 118-121.
CODEN: ITMZBJ. ISSN: 0303-6251.
FILE SEGMENT: BA; OLD
LANGUAGE: German
AU SCHMIDT J; **FRIEDRICHS G**; HEINMUELLER D; WACH J

L6 ANSWER 7 OF 22 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1987:222922 BIOSIS
DOCUMENT NUMBER: BR32:108796
TITLE: EFFECT OF WHR-2936 A NEW POSITIVE INOTROPIC AGENT IN GANGLIONIC-BETA BLOCKED DOGS.
AUTHOR(S): BARRETT J A; WOLTMANN R; KASIEWSKI C; **SWILLO R**; FAITH W C; PENDLETON R G
CORPORATE SOURCE: RORER CENTRAL RES., FORT WASHINGTON, PA.
SOURCE: 71ST ANNUAL MEETING OF THE FEDERATION OF AMERICAN SOCIETIES
FOR EXPERIMENTAL BIOLOGY, WASHINGTON, D.C., USA, MARCH 29-APRIL 2, 1987. FED PROC, (1987) 46 (3), 372.
CODEN: FEPRA7. ISSN: 0014-9446.

DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English
AU BARRETT J A; WOLTMANN R; KASIEWSKI C; **SWILLO R**; FAITH W C; PENDLETON R G

L6 ANSWER 8 OF 22 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 81274324 MEDLINE
DOCUMENT NUMBER: 81274324 PubMed ID: 7268191
TITLE: The pharmacodynamics of bucainide (RHC G233): pharmacokinetic parameters and relationship between plasma levels and the effect on the electrocardiogram in the dog.
AUTHOR: Grebow P; Feeney W; Johnston M; Lettieri J; Li H; Magnien E; O'Brien P; **Swillo R**; Weinryb I; Wolf P; Marsiglia J C
SOURCE: RESEARCH COMMUNICATIONS IN CHEMICAL PATHOLOGY AND PHARMACOLOGY, (1981 Jun) 32 (3) 407-21. Journal code: 0244734. ISSN: 0034-5164.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198110
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19811014
AU Grebow P; Feeney W; Johnston M; Lettieri J; Li H; Magnien E; O'Brien P; **Swillo R**; Weinryb I; Wolf P; Marsiglia J C

L6 ANSWER 9 OF 22 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1981:297872 BIOSIS
DOCUMENT NUMBER: BA72:82856
TITLE: PHARMACODYNAMICS OF BUCAINIDE RHC-G-233 PHARMACO KINETIC PARAMETERS AND RELATIONSHIP BETWEEN PLASMA LEVELS AND THE EFFECT ON THE ELECTRO CARDIOGRAM IN THE DOG.
AUTHOR(S): GREBOW P; FEENEY W; JOHNSTON M; LETTIERI J; LI H; MAGNIEN E; O'BRIEN P; **SWILLO R**; WEINRYB I; WOLF P; MARSIGLIA J C
CORPORATE SOURCE: DEP. BIOCHEMICTRY, RESEARCH AND DEVELOPMENT DIVISION, TUCKAHOE, N.Y.
SOURCE: RES COMMUN CHEM PATHOL PHARMACOL, (1981) 32 (8), 407-422. CODEN: RCOCB8. ISSN: 0034-5164.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AU GREBOW P; FEENEY W; JOHNSTON M; LETTIERI J; LI H; MAGNIEN E; O'BRIEN P; **SWILLO R**; WEINRYB I; WOLF P; MARSIGLIA J C

L6 ANSWER 10 OF 22 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 81179806 MEDLINE
DOCUMENT NUMBER: 81179806 PubMed ID: 7223169
TITLE: [Determination of radicality of iliacal lymphonodectomy in Wertheim's radical operation (author's transl)]. Radikalitatsbestimmung der iliakalen Lymphonodektomie im Rahmen der Wertheimschen Radikaloperation.
AUTHOR: Leitsmann H; Pawlowitsch T; Bilek K; **Friedrichs G**
SOURCE: ZENTRALBLATT FUR GYNAKOLOGIE, (1981) 103 (1) 53-62. Journal code: 21820100R. ISSN: 0044-4197.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German

FILE SEGMENT: Priority Journals
ENTRY MONTH: 198106
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810613
AU Leitsmann H; Pawlowitsch T; Bilek K; **Friedrichs G**

L6 ANSWER 11 OF 22 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 81128485 MEDLINE
DOCUMENT NUMBER: 81128485 PubMed ID: 555141
TITLE: [Gastrointestinal fiberoptic endoscopy in geriatric patients--experience, results and indications (author's transl)].
Erfahrungen, Ergebnisse und Indikationen der Fibroosophagogastroskopie bei geriatrischen Patienten.
AUTHOR: **Friedrichs G**; Gartner C; Wagner S; Muhlich H E
SOURCE: ZFA. ZEITSCHRIFT FUR ALTERNSFORSCHUNG, (1979) 34 (5) 445-53.
Journal code: 7704731. ISSN: 0044-2224.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198104
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810424
AU **Friedrichs G**; Gartner C; Wagner S; Muhlich H E

L6 ANSWER 12 OF 22 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 79099348 MEDLINE
DOCUMENT NUMBER: 79099348 PubMed ID: 735258
TITLE: [The retothel sarcoma of the stomach].
Uber das Retothelsarkom des Magens.
AUTHOR: Gartner C; Schumann H J; **Friedrichs G**; Muhlich H E
SOURCE: ZEITSCHRIFT FUR DIE GESAMTE INNERE MEDIZIN UND IHRE GRENZGEBIETE, (1978 Nov 1) 33 (21) 806-9.
Journal code: 21730470R. ISSN: 0044-2542.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197903
ENTRY DATE: Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19790313
AU Gartner C; Schumann H J; **Friedrichs G**; Muhlich H E

L6 ANSWER 13 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1978:586568 CAPLUS
DOCUMENT NUMBER: 89:186568
TITLE: Beryllium-containing catalysts for the selective Fischer-Tropsch synthesis
AUTHOR(S): Hammer, H.; **Friedrichs, G.**
CORPORATE SOURCE: Inst. Brennstoffchem. Phys.-Chem. Verfahrenstech., Tech. Hochsch. Aachen, Aachen, Ger.
SOURCE: Erdoel Kohle, Erdgas, Petrochem. (1978), 31(8), 370
CODEN: EKEPAB; ISSN: 0014-0058
DOCUMENT TYPE: Journal
LANGUAGE: German

AU Hammer, H.; **Friedrichs, G.**

L6 ANSWER 14 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1980:523881 CAPLUS
DOCUMENT NUMBER: 93:123881
TITLE: Study of surface morphology and crystal structure of pure and silver- and lithium-doped zinc oxide layers
AUTHOR(S): Ostrowski, J.; Sadowski, J.; **Swillo, R.**; Zmija, J.
CORPORATE SOURCE: Wojskowa Akad. Tech., Warsaw, Pol.
SOURCE: Fiz. Cienkich Warstw, Ogolnopol. Symp., 2nd (1977), Meeting Date 1975, 216-23. Editor(s): Zdanowicz, Lidia. Panst. Wydawn. Nauk.-Wroclaw.: Wroclaw, Pol. CODEN: 43TXAZ
DOCUMENT TYPE: Conference
LANGUAGE: Polish
AU Ostrowski, J.; Sadowski, J.; **Swillo, R.**; Zmija, J.

L6 ANSWER 15 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1980:541083 CAPLUS
DOCUMENT NUMBER: 93:141083
TITLE: Effect of the type of substrate and its position in relation to the target on the morphology and structure of zinc oxide layers deposited by high-frequency sputtering
AUTHOR(S): Ostrowski, J.; Sadowski, J.; **Swillo, R.**; Zmija, J.
CORPORATE SOURCE: Wojskowa Akad. Tech., Warsaw, Pol.
SOURCE: Fiz. Cienkich Warstw, Ogolnopol. Symp., 2nd (1977), Meeting Date 1975, 209-15. Editor(s): Zdanowicz, Lidia. Panst. Wydawn. Nauk.-Wroclaw.: Wroclaw, Pol. CODEN: 43TXAZ
DOCUMENT TYPE: Conference
LANGUAGE: Polish
AU Ostrowski, J.; Sadowski, J.; **Swillo, R.**; Zmija, J.

L6 ANSWER 16 OF 22 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 77197890 MEDLINE
DOCUMENT NUMBER: 77197890 PubMed ID: 868195
TITLE: [Hematuria due to foreign bodies in a woman-patient with the Munchhausen syndrome]. Hamaturie durch Fremdkorper bei Patientin mit Munchhausen-Syndrom.
AUTHOR: **Friedrichs G**; Gartner C; Gartner S; Schoeppner H
SOURCE: ZEITSCHRIFT FUR DIE GESAMTE INNERE MEDIZIN UND IHRE GRENZGEBIETE, (1977 Mar 15) 32 (6) 149-50. Journal code: 21730470R. ISSN: 0044-2542.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197707
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19770723
AU **Friedrichs G**; Gartner C; Gartner S; Schoeppner H

L6 ANSWER 17 OF 22 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1970:58055 BIOSIS
DOCUMENT NUMBER: BR06:58055

TITLE: ANTI TUSSIVE ACTIVITY OF O 4 METHOXYPHENYLCARBAMOYL-3-DIETHYLAMINO PROPIOPHENONE OXIME HYDRO CHLORIDE.
AUTHOR(S): ROMANO D V; GLASSMAN J M; GERACI C; **SWILLO R**
SOURCE: Pharmacologist, (1969) 11 (2), 257.
CODEN: PHMCAA. ISSN: 0031-7004.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: Unavailable
AU ROMANO D V; GLASSMAN J M; GERACI C; **SWILLO R**

L6 ANSWER 18 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1941:8989 CAPLUS
DOCUMENT NUMBER: 35:8989
ORIGINAL REFERENCE NO.: 35:1450c
TITLE: Change in the reduction-oxidation potential of the potato tuber due to storage temperature
AUTHOR(S): **Friedrichs, G.**
SOURCE: Angew. Botan. (1939), 21, 374-82
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AU **Friedrichs, G.**

L6 ANSWER 19 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1934:1695 CAPLUS
DOCUMENT NUMBER: 28:1695
ORIGINAL REFERENCE NO.: 28:248c-e
TITLE: The determination of the adhesiveness of dusts to treated cereal seed grain in the supervision of cooperative disinfection plants
AUTHOR(S): **Friedrichs, G.**
SOURCE: Rev. Applied Mycol. (1933), 12, 558-9
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AU **Friedrichs, G.**

L6 ANSWER 20 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1934:1694 CAPLUS
DOCUMENT NUMBER: 28:1694
ORIGINAL REFERENCE NO.: 28:248c-e
TITLE: The determination of the adhesiveness of dusts to treated cereal seed grain in the supervision of cooperative disinfection plants
AUTHOR(S): **Friedrichs, G.**
SOURCE: Nachrichtenbl. Deut. Pflanzenschutzdienst (1933), 13, 25-7
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AU **Friedrichs, G.**

L6 ANSWER 21 OF 22 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1935:37589 CAPLUS
DOCUMENT NUMBER: 29:37589
ORIGINAL REFERENCE NO.: 29:4884f-g
TITLE: Control methods in dry seed-grain disinfection
AUTHOR(S): **Friedrichs, G.**
SOURCE: Nachr. deut. Pflanzenschutzdienst (1933), 13, 25-7
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AU **Friedrichs, G.**

L6 ANSWER 22 OF 22 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1928:25883 CAPLUS
DOCUMENT NUMBER: 22:25883
ORIGINAL REFERENCE NO.: 22:3014g
TITLE: Dry disinfection of grain with continuous dusting machines
AUTHOR(S): **Friedrichs, G.**
SOURCE: Fortschritte Landw. (1928), 3, 58-66
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AU **Friedrichs, G.**

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FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 13:21:34 ON 01 OCT 2002
L1 1544 S ETANERCEPT
L2 1 S P55TNFR:FC
L3 21 S FRIEDRICHS G /AU
L4 8 S SWILLO R /AU
L5 29 S L3 OR L4
L6 22 DUP REM L5 (7 DUPLICATES REMOVED)
L7 0 S L1 AND L6

=> d l2 ibib

L2 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:597821 CAPLUS
DOCUMENT NUMBER: 135:166023
TITLE: Method of treating or inhibiting cellular injury or cell death following an ischemic event
INVENTOR(S): Friedrichs, Gregory Scott; Swillo, Roberto Edward; Jow, Brian Hong-N.; Bridal, Terry Roy; Numann, Randal Edward; Warner, Linda Marie; Killar, Loran Marie
PATENT ASSIGNEE(S): American Home Products Corp., USA
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001058473	A1	20010816	WO 2001-US4048	20010208

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-501862 A 20000210
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

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=> s cellular (s) injury (s) tnf (s) antagonist

L8          46 CELLULAR (S) INJURY (S) TNF (S) ANTAGONIST

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     (FILE 'HOME' ENTERED AT 13:21:20 ON 01 OCT 2002)

     FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 13:21:34 ON 01 OCT 2002
L1          1544 S ETANERCEPT
L2             1 S P55TNFR:FC
L3            21 S FRIEDRICHS G /AU
L4             8 S SWILLO R /AU
L5            29 S L3 OR L4
L6            22 DUP REM L5 (7 DUPLICATES REMOVED)
L7             0 S L1 AND L6
L8            46 S CELLULAR (S) INJURY (S) TNF (S) ANTAGONIST

=> s l1 and l8

L9             2 L1 AND L8

=> dup rem l9

PROCESSING COMPLETED FOR L9
L10            2 DUP REM L9 (0 DUPLICATES REMOVED)

=> d l10


L10  ANSWER 1 OF 2  EMBASE  COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN   2002206471  EMBASE
TI   Tumor necrosis factor-.alpha. in cardiovascular biology and the potential
     role for anti-tumor necrosis factor-.alpha. therapy in heart disease.
AU   Sack M.N.
CS   M.N. Sack, Hatter Inst. for Cardiology Research, MRC Inter-Univ. Cape
     Heart Group, Univ. of Cape Town Medical School, Observatory 7925, South
     Africa. sack@capeheart.uct.ac.za
SO   Pharmacology and Therapeutics, (2002) 94/1-2 (123-135).
     Refs: 136
     ISSN: 0163-7258  CODEN: PHTHDT
PUI  S 0163-7258(02)00176-6
CY   United States
DT   Journal; General Review
FS   018     Cardiovascular Diseases and Cardiovascular Surgery
     026     Immunology, Serology and Transplantation
     030     Pharmacology
     037     Drug Literature Index
     038     Adverse Reactions Titles
LA   English
SL   English


=> d l10 total ibib


L10  ANSWER 1 OF 2  EMBASE  COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER:   2002206471  EMBASE
```

TITLE: Tumor necrosis factor-.alpha. in cardiovascular biology and
the potential role for anti-tumor necrosis factor-.alpha. therapy in heart disease.
AUTHOR: Sack M.N.
CORPORATE SOURCE: M.N. Sack, Hatter Inst. for Cardiology Research, MRC Inter-Univ. Cape Heart Group, Univ. of Cape Town Medical School, Observatory 7925, South Africa. sack@capeheart.uct.ac.za
SOURCE: Pharmacology and Therapeutics, (2002) 94/1-2 (123-135). Refs: 136
ISSN: 0163-7258 CODEN: PHTHDT
PUBLISHER IDENT.: S 0163-7258(02)00176-6
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

L10 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:597821 CAPLUS
DOCUMENT NUMBER: 135:166023
TITLE: Method of treating or inhibiting cellular injury or cell death following an ischemic event
INVENTOR(S): Friedrichs, Gregory Scott; Swillo, Roberto Edward; Jow, Brian Hong-N.; Bridal, Terry Roy; Numann, Randal Edward; Warner, Linda Marie; Killar, Loran Marie
PATENT ASSIGNEE(S): American Home Products Corp., USA
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001058473	A1	20010816	WO 2001-US4048	20010208

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-501862 A 20000210
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT

=> d his

(FILE 'HOME' ENTERED AT 13:21:20 ON 01 OCT 2002)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 13:21:34 ON 01 OCT 2002
L1 1544 S ETANERCEPT
L2 1 S P55TNFR:FC
L3 21 S FRIEDRICHS G /AU
L4 8 S SWILLO R /AU
L5 29 S L3 OR L4
L6 22 DUP REM L5 (7 DUPLICATES REMOVED)
L7 0 S L1 AND L6
L8 46 S CELLULAR (S) INJURY (S) TNF (S) ANTAGONIST
L9 2 S L1 AND L8
L10 2 DUP REM L9 (0 DUPLICATES REMOVED)

=> dup rem l8

PROCESSING COMPLETED FOR L8
L11 20 DUP REM L8 (26 DUPLICATES REMOVED)

=> d l11 total ibib kwic

L11 ANSWER 1 OF 20 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002325293 MEDLINE
DOCUMENT NUMBER: 22063282 PubMed ID: 11940570
TITLE: Lipopolysaccharide-mediated reactive oxygen species and signal transduction in the regulation of interleukin-1 gene expression.
AUTHOR: Hsu Hsien-Yeh; Wen Meng-Hsuan
CORPORATE SOURCE: Faculty of Medical Technology, Institute of Biotechnology in Medicine, National Yang-Ming University, 112 Taipei, Taiwan.. hyhsu@ym.edu.tw
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2002 Jun 21) 277 (25) 22131-9.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 20020618
Last Updated on STN: 20020720
Entered Medline: 20020719

AB Lipopolysaccharide (LPS) stimulates macrophages to release inflammatory cytokines, interleukin-1 beta (IL-1), and tumor necrosis factor (**TNF**). LPS-induced **TNF** suppresses scavenger receptor functions in macrophages (van Lenten, B. J., and Fogelman, A. M. (1992) J. Immunol. 148, 112-116), which is regulated by **TNF**-mediated protein kinases (Hsu, H. Y., and Twu, Y. C. (2000) J. Biol. Chem. 275, 41035-41048). To examine the molecular mechanism. . . activity to 60% and decreased p38 activity to the basal level, but JNK activity was induced 2-fold. Furthermore, the pharmacological **antagonists** LY294002, SB203580, curcumin, calphostin C, and PD98059 revealed the diverse roles of LPS-mediated protein kinases in pro-IL-1. On the other. . . IL-1 induction upon the antibacterial action of macrophages should provide a therapeutic strategy for aberrant inflammatory responses leading to severe **cellular injury** or concurrent multiorgan septic damage.

L11 ANSWER 2 OF 20 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2002149687 MEDLINE
DOCUMENT NUMBER: 21874120 PubMed ID: 11867742
TITLE: The antitumor histone deacetylase inhibitor suberoylanilide hydroxamic acid exhibits antiinflammatory properties via suppression of cytokines.
AUTHOR: Leoni Flavio; Zaliani Andrea; Bertolini Giorgio; Porro Giulia; Pagani Paolo; Pozzi Pietro; Dona Giancarlo; Fossati Gianluca; Sozzani Silvano; Azam Tania; Bufler Philip; Fantuzzi Giamila; Goncharov Igor; Kim Soo-Hyun; Pomerantz Benjamin J; Reznikov Leonid L; Siegmund Britta; Dinarello Charles A; Mascagni Paolo
CORPORATE SOURCE: Italfarmaco, SpA., 20092 Cinisello Balsamo, Italy.. f.leoni@italfarmaco.com
CONTRACT NUMBER: AI 15614 (NIAID)
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2002 Mar 5) 99 (5) 2995-3000. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020308
Last Updated on STN: 20020429
Entered Medline: 20020426

AB . . . production of proinflammatory cytokines in vivo and in vitro. A single oral administration of SAHA to mice dose-dependently reduced circulating **TNF**-alpha, IL-1-beta, IL-6, and IFN-gamma induced by lipopolysaccharide (LPS). Administration of SAHA also reduced hepatic **cellular injury** in mice following i.v. injection of Con A. SAHA inhibited nitric oxide release in mouse macrophages stimulated by the combination of **TNF**-alpha plus IFN-gamma. Human peripheral blood mononuclear cells stimulated with LPS in the presence of SAHA released less **TNF**-alpha, IL-1-beta, IL-12, and IFN-gamma (50% reduction at 100-200 nM). The production of IFN-gamma stimulated by IL-18 plus IL-12 was also. . . by SAHA (85% at 200 nM). However, SAHA did not affect LPS-induced synthesis of the IL-1-beta precursor, the IL-1 receptor **antagonist**, or the chemokine IL-8. In addition, IFN-gamma induced by anti-CD3 was not suppressed by SAHA. Steady-state mRNA levels for LPS-induced **TNF**-alpha and IFN-gamma in peripheral blood mononuclear cells were markedly decreased, whereas IL-8 and IL-1-beta mRNA levels were unaffected. Because SAHA. . .

L11 ANSWER 3 OF 20 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2002166831 MEDLINE
DOCUMENT NUMBER: 21866505 PubMed ID: 11877481
TITLE: Cysteinyl leukotrienes and uridine diphosphate induce cytokine generation by human mast cells through an interleukin 4-regulated pathway that is inhibited by leukotriene receptor antagonists.
AUTHOR: Mellor Elizabeth A; Austen K Frank; Boyce Joshua A
CORPORATE SOURCE: Division of Rheumatology, Immunology and Allergy, Brigham and Women's Hospital, Boston, MA 02115, USA.
CONTRACT NUMBER: AI-31599 (NIAID)
HL-36110 (NHLBI)

SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (2002 Mar 4) 195 (5) 583-92.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020320
Last Updated on STN: 20020404
Entered Medline: 20020402

AB . . . induces a calcium flux in response to cysteinyl leukotrienes (cys-LTs) and uridine diphosphate (UDP) that is blocked by cys-LT receptor **antagonists**. We speculated that this IL-4-dependent, receptor-mediated response to the cys-LTs and UDP might induce cytokine generation by hMCs without concomitant. . . d with IL-4 responded to UDP (1microM), LTC(4) (100 nM), and LTD(4) (100 nM) by producing IL-5, tumor necrosis factor (**TNF**)-alpha, and especially large quantities of macrophage inflammatory protein (MIP)-1beta de novo at 6 h, preceded by the induced expression of. . . cytokine production by the primed hMCs occurred without histamine release or PGD(2) generation and was inhibited by the CysLT1 receptor **antagonist** MK571. Additionally, pretreatment of hMCs with MK571 or with the cys-LT biosynthetic inhibitor MK886 decreased IL-5 and **TNF**-alpha production in response to IgE receptor cross-linkage, implying a positive feedback by endogenously produced cys-LTs. Cys-LTs and UDP thus orchestrate a novel, IL-4-regulated, non-IgE-dependent hMC activation for cytokine gene induction that could be initiated by microbes, **cellular injury**, or neurogenic or inflammatory signals; and this pathobiologic event would not be recognized in tissue studies where hMC activation is. . .

L11 ANSWER 4 OF 20 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2002281563 EMBASE
TITLE: The effect of antihistamine on endotoxin-induced acute lung injury.
AUTHOR: Jung B.H.; Koh Y.; Kim W.D.
CORPORATE SOURCE: Dr. Y. Koh, Division of Critical Care Medicine, Asan Medical Center, Univ. of Ulsan College of Medicine, 388-1, Pungnap Dong, Songpa-gu, Seoul 138-736, Korea, Republic of.
yskoh@amc.seoul.kr
SOURCE: Tuberculosis and Respiratory Diseases, (2002) 52/3 (219-229).
Refs: 26
ISSN: 0378-0066 CODEN: KHCHAM
COUNTRY: Korea, Republic of
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
005 General Pathology and Pathological Anatomy
015 Chest Diseases, Thoracic Surgery and Tuberculosis
030 Pharmacology
037 Drug Literature Index
LANGUAGE: Korean
SUMMARY LANGUAGE: English

AB Background: Sepsis-induced acute lung **injury** (ALI) is caused by many **cellular** and humoral mediators induced by an endotoxin. Histamine, which is widely distributed in the lungs and has been considered as. . . were infused intratracheally by normal saline, 2) an

endotoxin group, where lipopolysaccharide (LPS) was administered intratracheally 3) the H(2) receptor **antagonist**-treated group (H(2) group) and 4) the H(1) receptor **antagonist**-treated group (H(1) group), where H(2)-receptor blocker (ranitidine) and H(1)-receptor blocker (pyrilamine) were co-treated intravenously with the intratracheal administration of an. . . the lung lavage fluid, myeloporoxidase (MPO) activity in the lung tissue, the pathologic score and the total number of neutrophils, **TNF**-.alpha.-IL-I.beta. and IL-10 in lung lavage (BAL) fluid were measured in each group as the indices of lung **injury**. Result: Compared to the control group, the endotoxin group exhibited significant increases in all lung **injury** indices. Significant reductions in the endotoxin-mediated increases in lung leak index ($p<0.05$) were observed in both the H(1) and H(2). . . neutrophils in the BAL fluid in both the H(2) and H(1) groups compared to the endotoxin group. The increases in **TNF**-.alpha. IL-1.beta. and IL-10 concentrations in the BAL fluid observed in the endotoxin group were not reduced in the H(2) and. . . endotoxin via the H(2) receptor. However the attenuating mechanism may not be related to the pathogenesis of neutrophil dependent lung **injury**.

L11 ANSWER 5 OF 20 MEDLINE
ACCESSION NUMBER: 2002432921 IN-PROCESS
DOCUMENT NUMBER: 22179886 PubMed ID: 12191598
TITLE: Tumor necrosis factor-alpha in cardiovascular biology and the potential role for anti-tumor necrosis factor-alpha therapy in heart disease.
AUTHOR: Sack Michael
CORPORATE SOURCE: Hatter Institute for Cardiology Research and MRC Inter-University Cape Heart Group, University of Cape Town Medical School, Observatory, 7925, South Africa.
SOURCE: PHARMACOLOGY AND THERAPEUTICS, (2002 Apr-May) 94 (1-2) 123.
Journal code: 7905840. ISSN: 0163-7258.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20020823
Last Updated on STN: 20020823

AB The functional role of tumor necrosis factor (**TNF**)-alpha in the heart has been extensively studied over the last 15 years. Collectively, these studies have demonstrated that **TNF**-alpha has both diverse and potentially conflicting roles in cardiac function and pathology. These include beneficial effects, such as cardioprotection against ischemia, myocarditis, and pressure overload, as well as potentially adverse effects, such as the development of atherosclerosis, reperfusion **injury**, hypertrophy, and heart failure. **TNF**-alpha **antagonist** therapy recently has been demonstrated to be clinically applicable in inflammatory conditions, and clinical trials are currently in progress in the use of these agents in cardiovascular diseases. The scope for clinical applications of anti-**TNF**-alpha therapy in cardiovascular diseases is potentially extensive. Hence, this review has been undertaken to evaluate the cardiovascular effects of this pleiotropic cytokine and to evaluate the potential of targeting this cytokine in cardiovascular therapeutics. An overview of the **TNF**-alpha peptide and its associated signaling are described. This is followed by a discussion of the known roles of **TNF**-alpha in cardiac physiology

and in a diverse array of cardiac pathologies. Reference to experimental and clinical studies using anti-**TNF**-alpha therapies are described where applicable. The postulated role of **TNF**-alpha signaling concerning innate cardiac **cellular** processes that may have direct adaptive effects in the heart will be reviewed with respect to future research directions. Finally, the author postulates that attenuation of **TNF**-alpha biosynthesis in selected individuals will need to be tested if true benefits of this therapeutic approach are to be realized. . .

L11 ANSWER 6 OF 20 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2002206471 EMBASE
TITLE: Tumor necrosis factor-.alpha. in cardiovascular biology and the potential role for anti-tumor necrosis factor-.alpha. therapy in heart disease.
AUTHOR: Sack M.N.
CORPORATE SOURCE: M.N. Sack, Hatter Inst. for Cardiology Research, MRC Inter-Univ. Cape Heart Group, Univ. of Cape Town Medical School, Observatory 7925, South Africa. sack@capeheart.uct.ac.za
SOURCE: Pharmacology and Therapeutics, (2002) 94/1-2 (123-135). Refs: 136
ISSN: 0163-7258 CODEN: PHTHDT
PUBLISHER IDENT.: S 0163-7258(02)00176-6
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The functional role of tumor necrosis factor (**TNF**)-.alpha. in the heart has been extensively studied over the last 15 years. Collectively, these studies have demonstrated that **TNF**-.alpha. has both diverse and potentially conflicting roles in cardiac function and pathology. These include beneficial effects, such as cardioprotection against ischemia, myocarditis, and pressure overload, as well as potentially adverse effects, such as the development of atherosclerosis, reperfusion **injury**, hypertrophy, and heart failure. **TNF**-.alpha. **antagonist** therapy recently has been demonstrated to be clinically applicable in inflammatory conditions, and clinical trials are currently in progress in the use of these agents in cardiovascular diseases. The scope for clinical applications of anti-**TNF**-.alpha. therapy in cardiovascular diseases is potentially extensive. Hence, this review has been undertaken to evaluate the cardiovascular effects of this pleiotropic cytokine and to evaluate the potential of targeting this cytokine in cardiovascular therapeutics. An overview of the **TNF**-.alpha. peptide and its associated signaling are described. This is followed by a discussion of the known roles of **TNF**-.alpha. in cardiac physiology and in a diverse array of cardiac pathologies. Reference to experimental and clinical studies using anti-**TNF**-.alpha. therapies are described where applicable. The postulated role of **TNF**-.alpha. signaling concerning innate cardiac **cellular** processes that may have direct adaptive effects in the heart will be reviewed with respect to future research directions.

Finally, the author postulates that attenuation of **TNF**-.alpha. biosynthesis in selected individuals will need to be tested if true benefits of this therapeutic approach are to be realized. . .

L11 ANSWER 7 OF 20 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:597821 CAPLUS
DOCUMENT NUMBER: 135:166023
TITLE: Method of treating or inhibiting cellular injury or cell death following an ischemic event
INVENTOR(S): Friedrichs, Gregory Scott; Swillo, Roberto Edward; Jow, Brian Hong-N.; Bridal, Terry Roy; Numann, Randal Edward; Warner, Linda Marie; Killar, Loran Marie
PATENT ASSIGNEE(S): American Home Products Corp., USA
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001058473	A1	20010816	WO 2001-US4048	20010208

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-501862 A 20000210
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
FORMAT
AB This invention provides a method of treating or inhibiting **cellular injury** or cell death following an ischemic event, treating or inhibiting reperfusion **injury**, and reducing mortality following a myocardial infarction by providing therapy with a **TNF**.alpha. **antagonist**.

L11 ANSWER 8 OF 20 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2001664012 MEDLINE
DOCUMENT NUMBER: 21566533 PubMed ID: 11709803
TITLE: Role of angiotensin II in tubulointerstitial injury.
AUTHOR: Cao Z; Cooper M E
CORPORATE SOURCE: Department of Medicine, University of Melbourne, Austin, Australia.
SOURCE: SEMINARS IN NEPHROLOGY, (2001 Nov) 21 (6) 554-62. Ref: 55
Journal code: 8110298. ISSN: 0270-9295.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011119
Last Updated on STN: 20020123
Entered Medline: 20011221

AB The renin angiotensin system (RAS) has been implicated in tubulointerstitial **injury** in a range of clinical and experimental settings. Angiotensin II, the major effector molecule of the RAS, in addition to. . . influencing renal tubular and interstitial function and structure including regulation of multiple cytokines and chemokines, promoting infiltration of monocytes/macrophages, promoting **cellular** proliferation, and inducing apoptosis. Pathologic actions of angiotensin II lead to tubulointerstitial fibrosis and inflammation via a range of cytokines and chemokines including transforming growth factor (**TNF**)-beta1, osteopontin, tumor necrosis factor (**TNF**)-alpha, secreted protein acidic and rich in cysteine (SPARC), and RANTES (regulated on activation normal T-cell expression and secreted). Blockade of. . . of angiotensin II by an angiotensin-converting enzyme (ACE) inhibitor or angiotensin II receptor antagonism with an angiotensin type 1 receptor **antagonist** has been shown to attenuate tubulointerstitial **injury** and reduce expression of cytokines and matrix proteins. The role of angiotensin II in tubulointerstitial fibrosis and inflammation is addressed. . .

L11 ANSWER 9 OF 20 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 2000219266 MEDLINE
DOCUMENT NUMBER: 20219266 PubMed ID: 10754324
TITLE: Cytokine-stimulated, but not HIV-infected, human monocyte-derived macrophages produce neurotoxic levels of l -cysteine.
AUTHOR: Yeh M W; Kaul M; Zheng J; Nottet H S; Thylin M; Gendelman H E; Lipton S A
CORPORATE SOURCE: Cerebrovascular and Neuroscience Research Institute, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115, USA.
CONTRACT NUMBER: P01HD29587 (NICHD)
R01EY09024 (NEI)
R01MH58164 (NIMH)
+
SOURCE: JOURNAL OF IMMUNOLOGY, (2000 Apr 15) 164 (8) 4265-70. Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; AIDS
ENTRY MONTH: 200005
ENTRY DATE: Entered STN: 20000518
Last Updated on STN: 20000518
Entered Medline: 20000509

AB . . . to infection of the brain with HIV. The cognitive manifestations have been termed HIV-associated dementia. The mechanisms underlying HIV-associated neuronal **injury** are incompletely understood, but various studies have confirmed the release of neurotoxins by macrophages/microglia infected with HIV-1 or stimulated by. . . possibility that l -cysteine, a neurotoxin acting at the N-methyl-d -aspartate subtype of glutamate receptor, could contribute to HIV-associated neuronal **injury**. Picomolar concentrations of gp120 were found to stimulate cysteine release from human monocyte-derived macrophages (hMDM) in amounts sufficient to injure cultured rat

cerebrocortical neurons. **TNF**-alpha and IL-1beta, known to be increased in HIV-encephalitic brains, as well as a **cellular** product of cytokine stimulation, ceramide, were also shown to induce release of cysteine from hMDM in a dose-dependent manner. A **TNF** -alpha-neutralizing Ab and an IL-1betaR **antagonist** partially blocked gp120-induced cysteine release, suggesting that these cytokines may mediate the actions of gp120. Interestingly, hMDM infected with HIV-1 produced significantly less cysteine than uninfected cells following stimulation with **TNF**-alpha. Our findings imply that cysteine may play a role in the pathogenesis of neuronal **injury** in HIV-associated dementia due to its release from immune-activated macrophages but not virus-infected macrophages. Such uninfected cells comprise the vast. . .

L11 ANSWER 10 OF 20 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 2000397317 MEDLINE
DOCUMENT NUMBER: 20359519 PubMed ID: 10899957
TITLE: Effects of nitrobenzylthioinosine on neuronal injury, adenosine levels, and adenosine receptor activity in rat forebrain ischemia.
AUTHOR: Parkinson F E; Zhang Y W; Shepel P N; Greenway S C; Peeling J; Geiger J D
CORPORATE SOURCE: Department of Pharmacology and Therapeutics, University of Manitoba, Winnipeg, Canada.. fiona_parkinson@umanitoba.ca
SOURCE: JOURNAL OF NEUROCHEMISTRY, (2000 Aug) 75 (2) 795-802. Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000824
Last Updated on STN: 20000824
Entered Medline: 20000817

AB . . . (NBMPR), a selective and potent inhibitor of one adenosine transporter subtype termed ENT1, or es, can protect against ischemic neuronal **injury** by enhancing adenosine levels and potentiating adenosine receptor-mediated effects, including attenuation of the **cellular** production and release of tumor necrosis factor-alpha (**TNF**-alpha). In rats, the phosphorylated prodrug form of NBMPR, NBMPR-phosphate, or saline was administered by intracerebroventricular injection 30 min before forebrain. . . levels; however, this treatment tended to increase adenosine levels in all brain regions at 7 min postreperfusion. Ischemia-induced expression of **TNF**-alpha was not altered by NBMPR-P treatment, and the nonselective adenosine receptor **antagonist** 8-(p-sulfophenyl) theophylline did not abolish the neuroprotective effects of NBMPR-P treatment. These data indicate that NBMPR can protect CA1 pyramidal neurons from ischemic death without statistically significant effects on adenosine levels or adenosine receptor-mediated inhibition of the proinflammatory cytokine **TNF** -alpha.

L11 ANSWER 11 OF 20 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 2000389763 MEDLINE
DOCUMENT NUMBER: 20374225 PubMed ID: 10919572
TITLE: Neutralization of tumor necrosis factor-alpha action delays but does not prevent lung injury induced by alloreactive T helper 1 cells.
AUTHOR: Clark J G; Mandac J B; Dixon A E; Martin P J; Hackman R C;

Madtes D K
CORPORATE SOURCE: Fred Hutchinson Cancer Research Center, Department of Medicine, University of Washington School of Medicine, Seattle 98109-1024, USA.. jclark@fhcrc.org
CONTRACT NUMBER: HL 30542 (NHLBI)
HL 49401 (NHLBI)
HL 55200 (NHLBI)
SOURCE: TRANSPLANTATION, (2000 Jul 15) 70 (1) 39-43.
Journal code: 0132144. ISSN: 0041-1337.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000818
Last Updated on STN: 20000818
Entered Medline: 20000810

AB BACKGROUND: Lung **injury** occurs frequently after allogeneic bone marrow transplantation in association with graft-versus-host disease, an immune response that involves both **cellular** and cytokine components. In a murine model, we recently showed that cloned alloreactive T helper (Th)1 cells can cause lung **injury** associated with increased production of tumor necrosis factor (**TNF**)-alpha by alveolar macrophages (J Immunol 1998; 161: 1913). METHODS: To evaluate the role of **TNF**-alpha in this model, we injected in vitro-activated Th1 cells into the following: (1) recipients deficient in receptors for **TNF**; (2) C57BL/6 control mice; (3) C57BL/6 mice, pretreated with soluble TNFRIIFc (a dimorphic high-affinity **TNF antagonist**); (4) mice expressing TNFRIIFc transgene under control of the surfactant apoprotein C promoter (SPCTNFRIIFc); and (5) wild-type littermate controls (C57BL/6). . . any of the experimental groups. CONCLUSIONS: We conclude that lung inflammation induced by Th1 cells may be only delayed when **TNF**-alpha action is blocked. The persistence of abnormalities indicates that other proinflammatory pathways are involved in **injury** caused by these cells.

L11 ANSWER 12 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:134337 BIOSIS
DOCUMENT NUMBER: PREV200100134337
TITLE: Differential physiological and pharmacological neuroimmune outcomes in rat models of neuropathic and radicular pain.
AUTHOR(S): DeLeo, J. A. (1); Arruda, J. L.; Hunt, J.; Rutkowski, M. D.; Sweitzer, S.; Winkelstein, B. A.; Wynkoop, T.
CORPORATE SOURCE: (1) Dartmouth Med Sch, Lebanon, NH USA
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-733.1. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience
. ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

AB. . . neuropathic and radicular pain. We have demonstrated increased spinal proinflammatory cytokines and glial activation following peripheral nerve or lumbar root **injuries** in the rat that result in pain behaviors suggestive of neuropathy or radiculopathy, respectively. In the

present study, we directly. . . hyperalgesia was significantly greater in the radiculopathy model as compared with the neuropathy model. An intrathecal cocktail of IL-1 receptor **antagonist** and soluble **TNF** receptor significantly attenuated allodynia in the neuropathy model but did not alter allodynia after the lumbar root **injury**. Immunohistochemistry for glial activation, cytokines, **cellular** adhesion molecules, CD+4 and Major Histocompatibility Complex class II was performed on L4-L5 spinal levels in all rats. Although there. . . model evoked a greater spinal neuroinflammatory response. These findings support the hypothesis that pain behaviors following L5 nerve or root **injury** may have a similar, albeit not identical, neuroimmune etiology.

L11 ANSWER 13 OF 20 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 97474356 MEDLINE
DOCUMENT NUMBER: 97474356 PubMed ID: 9335380
TITLE: Macrophages promote prosclerotic responses in cultured rat mesangial cells: a mechanism for the initiation of glomerulosclerosis.
AUTHOR: Pawluczyk I Z; Harris K P
CORPORATE SOURCE: Department of Nephrology, Leicester General Hospital, England, United Kingdom.
SOURCE: JOURNAL OF THE AMERICAN SOCIETY OF NEPHROLOGY, (1997 Oct) 8 (10) 1525-36.
Journal code: 9013836. ISSN: 1046-6673.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971121
AB Glomerulosclerosis is the final outcome of a number of different causes of glomerular **injury**, during which the structures of the glomerulus are obliterated by extracellular matrix. Accumulating evidence suggests that infiltrating macrophages play a pivotal role in the progression to glomerulosclerosis. The present study defines the role played by macrophages at both **cellular** and molecular levels in the initiation of the sclerotic process in cultured rat mesangial cells. Macrophage-conditioned medium (MPCM) generated from. . . both transin and tissue inhibitor of metalloproteinase-1 gene transcription. Transforming growth factor (TGF) beta1, platelet-derived growth factor, tumor necrosis factor (**TNF**) alpha, or interleukin (IL)-1beta could not be detected in the MPCM per se; however, TGFbeta1 and platelet-derived growth factor AB. . . and 5.7 +/- 1.2-fold [P < 0.004], respectively). Incubation of MPCM with either neutralizing antibody or the growth factor receptor **antagonist** suramin demonstrated that TGFbeta1 played a significant, although minor, role in MPCM-stimulated fibronectin production. In conclusion, this study provides compelling. . .

L11 ANSWER 14 OF 20 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 97260155 MEDLINE
DOCUMENT NUMBER: 97260155 PubMed ID: 9106250

TITLE: Neurotrophins and their receptors in nerve injury and repair.
AUTHOR: Ebadi M; Bashir R M; Heidrick M L; Hamada F M; Refaey H E; Hamed A; Helal G; Baxi M D; Cerutis D R; Lassi N K
CORPORATE SOURCE: Department of Pharmacology, University of Nebraska College of Medicine, Omaha 68198-6260, USA.
CONTRACT NUMBER: NS34566 (NINDS)
SOURCE: NEUROCHEMISTRY INTERNATIONAL, (1997 Apr-May) 30 (4-5) 347-74. Ref: 240
Journal code: 8006959. ISSN: 0197-0186.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970805
Last Updated on STN: 19980206
Entered Medline: 19970724

AB . . . IL-I beta, ILIra and IL-2-IL-15), chemokines (IL-8/ NAP-I, NAP-2,
MIP-I alpha and beta, MCAF/MCP-1, MGSA and RANTES), tumor necrosis factors
(**TNF**-alpha and **TNF**-beta), interferons (INF-alpha, beta
and gamma), colony stimulating factors (G-CSF, M-CSF, GM-CSF, IL-3 and
some of the other ILs), growth factors. . . nervous system (PNS). The
neurotrophins are currently under investigation as therapeutic agents for
the treatment of neurodegenerative disorders and nerve **injury**
either individually or in combination with other trophic factors such as
ciliary neurotrophic factor (CNTF) or fibroblast growth factor (FGF).. .
. transduction pathways. These include the ras-dependent pathway utilized
by trk to mediate neurotrophin effects such as survival and
differentiation. Indeed, **cellular** diversity in the nervous
system evolves from the concerted processes of cell proliferation,
differentiation, migration, survival, and synapse formation. Neural. .
.
response, including hypophagia and sleep. Cytokine production has been
detected within the central nervous system as a result of brain
injury, following stab wound to the brain, during viral and
bacterial infections (AIDS and meningitis), and in neurodegenerative
processes (multiple sclerosis and Alzheimer's disease). Novel cytokine
therapies, such as anticytokine antibodies or specific receptor
antagonists acting on the cytokine network may provide an
optimistic feature for treatment of multiple sclerosis and other diseases
in which. . .

L11 ANSWER 15 OF 20 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 1998036726 MEDLINE
DOCUMENT NUMBER: 98036726 PubMed ID: 9369986
TITLE: Inflammatory gene expression in cerebral ischemia and trauma. Potential new therapeutic targets.
AUTHOR: Feuerstein G Z; Wang X; Barone F C
CORPORATE SOURCE: Department of Cardiovascular Pharmacology, SmithKline Beecham Pharmaceuticals, King of Prussia, Pennsylvania 19406, USA.. giora_z_feuerstein@sbphrd.com
SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1997 Oct 15) 825 179-93. Ref: 104
Journal code: 7506858. ISSN: 0077-8923.
PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 20000303
Entered Medline: 19971209

AB . . . chemokines and endothelial-leukocyte adhesion molecules in the brain follow soon after the ischemic insult and at a time when the **cellular** component is evolving. The significance of the inflammatory response to brain ischemia is not fully understood. Evidence is emerging in. . . brain damage. This evidence includes: 1) the capacity of cytokines to exacerbate brain damage; 2) the capacity of specific cytokine **antagonists** such as IL-1ra to reduce ischemic brain damage; 3) that depletion of circulating neutrophils reduces ischemic brain **injury**; 4) and that **antagonists** of the endothelial-leukocyte adhesion interactions (e.g., anti-ICAM-1) reduce ischemic brain **injury**. However, it should be kept in mind that cytokines were also argued to provide beneficial effects in brain **injury** as inferred from studies with **TNF**-receptor knock-out mice (p55 and p75 knock-out), which display increased sensitivity to brain ischemia, and the capacity of IL-1 to elicit. . .

L11 ANSWER 16 OF 20 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 97397009 MEDLINE
DOCUMENT NUMBER: 97397009 PubMed ID: 9253161
TITLE: Effects of retinoids on the production of tumour necrosis factor-alpha and nitric oxide by lipopolysaccharide-stimulated rat Kupffer cells in vitro: evidence for participation of retinoid X receptor signalling pathway.
AUTHOR: Motomura K; Sakai H; Isobe H; Nawata H
CORPORATE SOURCE: Third Department of Internal Medicine, Faculty of Medicine,
Kyushu University, Fukuoka, Japan.
SOURCE: CELL BIOCHEMISTRY AND FUNCTION, (1997 Jun) 15 (2) 95-101.
Journal code: 8305874. ISSN: 0263-6484.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19970916
Last Updated on STN: 19970916
Entered Medline: 19970904

AB Kupffer cells play important roles in the development of liver **injury** by producing cytokines and free radicals. In consequence inhibition of these inflammatory mediators will be one of the targets for treating liver diseases. Retinoids modulate a wide variety of functions of monocytes/macrophages. **Cellular** effects of retinoids are mediated by two families of nuclear receptors, retinoic acid receptors (RARs) and retinoid X receptors (RXRs). We examined the effects of several kinds of natural and synthetic retinoids on the production of tumour necrosis factor-alpha (**TNF**-alpha) and nitric oxide (NO) by LPS-stimulated rat Kupffer cells in vitro. Of the various retinoids tested, 9-cis-retinoic acid (9-cis-RA) and Ro 13-6307 which are agonists

of both RARs and RXRs, suppressed the production of **TNF**-alpha and NO in a concentration-dependent fashion, whereas three types of RAR-selective agonists, Ro 13-7410, Ro 40-6055 and Ro 19-0645 did not show any effect. Furthermore, the RAR alpha **antagonist**, Ro 41-5253, did not prevent the effects induced by 9-cis-RA. The results suggest that these effects of 9-cis RA and. . .

L11 ANSWER 17 OF 20 MEDLINE DUPLICATE 12
ACCESSION NUMBER: 97204116 MEDLINE
DOCUMENT NUMBER: 97204116 PubMed ID: 9051688
TITLE: Involvement of tumor necrosis factor-alpha, interleukin-1 beta, interleukin-8, and interleukin-1 receptor antagonist in acute lung injury caused by local Shwartzman reaction.
AUTHOR: Imamura S; Matsukawa A; Ohkawara S; Kagayama M; Yoshinaga M
CORPORATE SOURCE: Department of Pathology, Kumamoto University School of Medicine, Japan.
SOURCE: PATHOLOGY INTERNATIONAL, (1997 Jan) 47 (1) 16-24. Journal code: 9431380. ISSN: 1320-5463.
PUB. COUNTRY: Australia
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970523
Last Updated on STN: 19970523
Entered Medline: 19970513

AB A local Shwartzman reaction (LSR) was prepared in rabbit lung as a model of acute lung **injury**. To induce LSR, intratracheal injection of lipopolysaccharide (LPS) 10 micrograms into the lower lobe of the right lung, followed 24. . . findings showed diffuse interstitial widening, intra-alveolar leukocyte infiltration with hemorrhage, and alveolar exudate formation. The production of tumor necrosis factor-alpha (**TNF**-alpha), interleukin-1 beta (IL-1 beta), interleukin-8 (IL-8), and IL-1 receptor **antagonist** (IL-1 Ra) in the lung was analyzed. **TNF**-alpha first elevated and peaked at 0.5 h (66.5 +/- 16.7 ng/g.lung), subsequently, IL-1 beta and IL-8 increased and peaked at. . . h, 1.6 +/- 0.1 micrograms/g.lung), and a large concentration of IL-1Ra was sustained for 48 h. Immunohistochemistry showed that the **cellular** source of these cytokines was alveolar macrophages and infiltrating neutrophils. Thus, disclosing the kinetics of the generation of cytokines led to a better understanding of their roles, namely **TNF**-alpha as an initiator, IL-1 and IL-8 as amplifier and effector, and IL-1Ra as regulator of the intensity of acute inflammation.

L11 ANSWER 18 OF 20 MEDLINE DUPLICATE 13
ACCESSION NUMBER: 96247662 MEDLINE
DOCUMENT NUMBER: 96247662 PubMed ID: 8666814
TITLE: Adenosine enhances IL-10 secretion by human monocytes.
AUTHOR: Le Moine O; Stordeur P; Schandene L; Marchant A; de Groote D; Goldman M; Deviere J
CORPORATE SOURCE: Department of Gastroenterology, Erasme Hospitol, Free University of Brussels, Belgium.
SOURCE: JOURNAL OF IMMUNOLOGY, (1996 Jun 1) 156 (11) 4408-14. Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199608

ENTRY DATE: Entered STN: 19960819
Last Updated on STN: 19960819
Entered Medline: 19960808

AB . . . effects on the production of IL-10 by human monocytes were presently investigated. Pre-incubation with adenosine dose-dependently enhanced IL-10 release by **TNF** stimulated human monocytes (+29, +58, and +116% at 1, 10, and 100 muM, respectively.) Adenosine also significantly enhanced IL-10 production after hydrogen peroxide and LPS stimulation and dose-dependently inhibited **TNF** secretion. Pre-incubation was not mandatory to achieve these effects, since addition of adenosine at the time of or 30 min after the stimulus led to the same results. Blocking IL-10 with anti-IL-10 mAbs partially restored adenosine-induced **TNF** inhibition. The enhanced IL-10 production was not observed when cells were preincubated with adenosine A1 or A2 receptor agonists (R-phenylisopropyladenosine, 5'-N-ethylcarboxamido-adenosine, and 2-chloroadenosine) and was not affected by pretreatment with theophyllin, an **antagonist** of both A1 and A2 receptors, or with dipyridamole, an inhibitor of adenosine **cellular** uptake. In conclusion, adenosine, in the submillimolar concentration range, increases IL-10 secretion by stimulated monocytes. This phenomenon participates in **TNF** inhibition, a known property of adenosine, but is not mediated through the occupancy of A1 or A2 receptors. This may represent a novel antiinflammatory property of adenosine by which it could modulate inflammation and limit ischemia-reperfusion **injury**.

L11 ANSWER 19 OF 20 MEDLINE DUPLICATE 14
ACCESSION NUMBER: 95217968 MEDLINE
DOCUMENT NUMBER: 95217968 PubMed ID: 7703308
TITLE: Effects of therapy with interleukin-1 receptor antagonist on pulmonary cytokine expression following hemorrhage and resuscitation.
AUTHOR: Abraham E; Allbee J
CORPORATE SOURCE: Division of Pulmonary Sciences and Critical Care Medicine, University of Colorado Health Sciences Center, Denver.
CONTRACT NUMBER: GM39102 (NIGMS)
SOURCE: LYMPHOKINE AND CYTOKINE RESEARCH, (1994 Dec) 13 (6) 343-7. Journal code: 9107882. ISSN: 1056-5477.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199505
ENTRY DATE: Entered STN: 19950518
Last Updated on STN: 19950518
Entered Medline: 19950511

AB Acute lung **injury** frequently develops following hemorrhage and is characterized by increased proinflammatory cytokine levels and massive neutrophil accumulation in the lung. Blood. . . and IL-1 beta mRNA expression among pulmonary cell populations. To examine the role of IL-1 in producing acute inflammatory lung **injury** after hemorrhage, we treated mice following hemorrhage and resuscitation with recombinant interleukin-1 receptor **antagonist** (IL-1Ra), a competitive inhibitor of the actions of IL-1. Therapy with IL-1Ra prevented the posthemorrhage increases in pulmonary **TNF**-alpha levels normally found after blood loss. Administration of IL-1Ra also diminished the increases in IL-1 beta and IL-6 mRNA levels that occur in the lungs following hemorrhage. However, the amounts of **TNF**-alpha and IFN-gamma mRNA among intraparenchymal pulmonary mononuclear cells remained elevated after hemorrhage despite therapy with IL-1Ra. These results

indicate that. . . . period is capable of normalizing the expression of some, but not all, of the proinflammatory cytokines whose production among pulmonary **cellular** populations is increased by blood loss.

L11 ANSWER 20 OF 20 MEDLINE DUPLICATE 15
ACCESSION NUMBER: 93322117 MEDLINE
DOCUMENT NUMBER: 93322117 PubMed ID: 8330929
TITLE: Killing of endothelial cells and release of arachidonic acid. Synergistic effects among hydrogen peroxide, membrane-damaging agents, cationic substances, and proteinases and their modulation by inhibitors.
AUTHOR: Ginsburg I; Mitra R S; Gibbs D F; Varani J; Kohen R
CORPORATE SOURCE: Department of Oral Biology, Hebrew University-Hadassah School of Medicine, Jerusalem, Israel.
CONTRACT NUMBER: GM-29507 (NIGMS)
HL-31963 (NHLBI)
SOURCE: INFLAMMATION, (1993 Jun) 17 (3) 295-319.
Journal code: 7600105. ISSN: 0360-3997.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199308
ENTRY DATE: Entered STN: 19930826
Last Updated on STN: 20000303
Entered Medline: 19930818

AB . . . with tannic acid and by extracts of tea, but less so by serum. On the other hand, neither deferoxamine, HClO, **TNF**, nor GTP gamma S had any modulating effects on the synergistic cell killing. EC exposed either to 6-deoxyglucose, puromycin, or. . . . scavengers of H2O2, by proteinase inhibitors, by cationic agents, by HClO, by tannic acid, and by quinacrin. We suggest that **cellular injury** induced in inflammatory and infectious sites might be the result of synergistic effects among leukocyte-derived oxidants, lysosomal hydrolases, cytotoxic cationic. . . . be also achieved following attack by leukocyte-derived agonists on dead cells. It is proposed that treatment by "cocktails" of adequate **antagonists** might be beneficial to protect against **cellular injury** in vivo.

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